GASTROENTEROLOGY, PART II

The patient who has diarrhea following a heavy meal consisting of shellfish and seafood, so the liver enzymes go up, and they ask you again what is the organism? Diarrhea following seafood plus liver dysfunctions. GI, Part II. The liver dysfunctions following ingestion of seafood equals Hepatitis A. How many people knew that? Three people, four, five. Liver dysfunction following seafood ingestion, in this case shellfish, is Hepatitis A. If you go to a prostitute, you always get Hepatitis B, and if you’re a drug abuser, you’ll get Hepatitis C. That’s why A, B, C. And if you go to a prostitute and use drugs and at the same time eat shellfish, [Laughter] you will get Hepatitis A, B, and C. [Laughter] Wholesale. [Laughter]

Question 2?

A 52-year-old male, CEO of a well-respected firm while in Thailand on a business trip happens to have a business deal with a prostitute. He comes back to the U.S. with jaundice. Three weeks before taking his trip, he claims to have had a vaccination for Hepatitis B. Liver enzymes are over 3,000 units. What is the possible diagnosis?

Okay. How many people think it’s B, like we said, Hepatitis B? How many people? Half? First of all, A and E are caught at a young age, and A and E are caught in India. I had E myself when I was 15 years old, but D occurs in drug addicts in patients who already have Hepatitis B. Sexual transmission most often occurs with B, and also fulminant disease occurs most often with B, so the answer here is B. It doesn’t matter whether you have the shots or no shots because if you had the shot, you don’t know if it was the antibody, and if you had the shots a long time ago, maybe the antibody went away, so shots or no shots, you are not going to go to a prostitute. [Laughter] In the exam, when the patient goes to a prostitute, it is Hepatitis B unless proven otherwise. Okay? But that’s the whole idea of the question they keep bringing.

Number 3?

Which test of Hepatitis B indicates maximum infectivity?

The red part here, this red part inside is the DNA, so that will be the most infectious, the innermost. The thing is we don’t do anti-DNA antibody for Hepatitis B as a rule of practice. That’s done in a research lab, so the next one is the yellow one, which contains Hepatitis core antigen and e-antigen, so the antibody against them would be next infectious after DNA, and there is no such thing as we don’t do core antigen in practice. It’s not available. So what is available to use is Hepatitis e-antigen, so the answer has to be on the exam Hepatitis e-antigen. Surface antigen also is infective, but it’s the least infective of the three, so the way the question was given, the answer is Hepatitis e-antigen.

In Hepatitis B serology, first you get these antigens. The DNA, e-antigen, surface antigen, all three, and as soon as you get going, you get ALT going up, and following that you start making the antibodies to each one of them. Eventually because of all these three antibodies, you do develop the immunity, and then the antigens disappear and you become immune here. If you are sure that you have all these and none of these then, yes, you could go to the prostitute, no problem. [Laughter] But you don’t know which patient is in this area, in this location.

So the tests we do for Hepatitis B or for A, B, and C, we said already e-antigen for Hepatitis B indicates infectious, and when you measure IgM of Hepatitis A, that will indicate
acute disease. IgG against A means that the infection has resolved and permanent immunity, usually permanent, has developed, which usually occurs in childhood, especially in other countries.

If you have surface antigen positive, that means either you have acute Hepatitis B or you have chronic Hepatitis B. If the antibody is present against Hepatitis B, that will indicate immunity. The core antigen in Hepatitis B is represented as small c, not capital C. Capital C is for Hepatitis C. The core antigen is a small c, is for Hepatitis B core antigen, and, of course, Hepatitis E antibody is against the e-antigen. That means you are not infectious anymore.

Whenever anti-c antibody is positive, you have to confirm with RNA PCR, so normally when you are in your hospital, Hepatitis C antibody, you are getting very rough, very non-specific test, which if positive you should never tell the patient that you’re Hepatitis C unless you have confirmed with RNA PCR because in many conditions when Hepatitis C is not present, the anti-HCV can be positive, so you never, ever—although you do the serology first for Hepatitis C, if it’s positive, you’d repeat the test with RNA PCR to confirm the presence of Hepatitis C. Otherwise you don’t tell the patient until that time.

And the natural history of Hepatitis B, you notice 95% recovery. Only a very few get persistent infection, so it’s not a bad disease. It’s okay. [Laughter] And you compare that with Hepatitis C, oh, my God. Almost everybody becomes chronic. See? Look at that. Fifty-five percent, [inaudible], become chronic. Fifty percent? My God. You get Hepatitis C, 50% chance of being chronic, and therefore out of that half will get liver cirrhosis. It’s an awful disease to have, and the beauty is in Hepatitis C you can have normal liver enzymes, so you don’t even know that you have it. Loads of people walking around with Hepatitis C, so that’s the difference between Hepatitis B and C.

So where are we now? You’re asked to do three questions in one minute.

[Three questions answered]

Okay, right answers first. See if you are correct. So Hepatitis C RNA by PCR, that is the test of choice. Okay, we did that. No. 2, the NIH Consensus Conference recommended not treating patients with normal liver enzymes, and out of the three genotypes, Genotype 1 is the one which is present in most of the United States, and this is the one which is resistant strain. Very few people have Genotype 2 and 3, where you can induce sustained response rate with the treatment ribavirin and interferon, but genotyping is done to find out what kind of—whether you could respond to treatment properly or not, so viral quantitation is very important, specifically if you’re going to start the treatment, and genotyping tells you whether you will get a response or not, although in practice lots of doctors, gastroenterologists, will give treatment with normal liver enzymes. For your purpose, for internal medicine Board exam, you will follow this. If the liver enzymes are normal, do not treat. Who will respond? Usually genotype other than Type 1, usually with a viral load less than 2 million, age less than 35, female sex, and either no or mild portal fibrosis, so Question No. 3, the answer is A because of that.

The problem with interferon is that it has a lot of side effects. Everybody gets flu, some people go crazy, so it’s not an easy drug to give, and on stopping the drug lots of people get decompensation. It’s not 100%. The Board is very much interested in knowing the contraindications for the use of interferon, and out of these four contraindications the one they keep asking in the exam is decompensated liver disease. As a matter of fact, there is no other question in the exam where one of the answers is decompensated liver disease. That’s why I want you to read the answers first. I want you to read the last line of the question and the answers first. Suppose among the answers you happen to see ‘decompensated liver disease.’ Take it. You don’t need to read the questions. [Laughter] There’s no other question with that kind of answer. It’s a very super-specific thing. Five questions, two minutes.
Day One

[Five questions answered]

Question No. 1, if you have a patient with periarteritis nodosa, you must remember that Hepatitis B can lead to periarteritis nodosa in some people, if somebody has a past history of Hepatitis B. Now he comes to you with vasculitis. That vasculitis is due to Hepatitis B. That’s a Board question. That’s why I’m putting it here for you. These are the extra-hepatic manifestations of Hepatitis B. To be more specific on vasculitis, the Board question out of all this is only one, actually, so you must know. A patient comes to you with pain in the right upper quadrant, so you think that it’s gallbladder, so you order an ultrasound. Ultrasound says there is no stone in the gallbladder, doctor, but there is edema of the wall of the gallbladder, so the patient has pain, the patient has edema of the wall, so you think of acalculous cholecystitis.

Mostly it is stone which causes cholecystitis, but sometimes acalculous cholecystitis, which is more dangerous than the stone, calculous cholecystitis, is the cause, and patient will have pain in the right upper quadrant. It occurs in old people from nursing homes all the time. They’re lying there with pain in the right upper quadrant and nobody knows because they don’t complain, so you do their blood count. Their WBC count is high, and then they’ve get a fever, and then they get sepsis, and then they die. What happened was the patient, because of old age and some other conditions and ischemia all over the body because of arteriosclerotic disease, they get ischemic vasculitis in the right upper quadrant involving the gallbladder, so that’s one scenario.

The other scenario is this. In a younger person, Hepatitis B in the past, right upper quadrant, gallbladder showing edema of the wall also is acalculous cholecystitis in a younger person with a past history of Hepatitis B, so you do it. What is your next step? The Board will ask you, doctor, what do you want to do next? Obviously you want to do a HIDA scan to see if it is a problem in the gallbladder or if it’s in the bile duct, so you do a HIDA scan, and the HIDA scan is positive by indicating, but it did not show up when they put the dye in, indicating the gallbladder is not working. If the gallbladder is not working, the patient has acute cholecystitis, but there was no stone, but this is called acalculous acute cholecystitis, in this case due to past Hepatitis B, and that is the whole question. The one in the nursing home you will see in your practice all the time. I have seen so many times. They don’t ask that in the exam, but this one they do, so acalculous cholecystitis due to vasculitis, which is due to Hepatitis B, which is due to you went to the prostitute. [Laughter] Simple as that. And this is what it is, periarteritis nodosa. See that node on the blood vessel? And you took a biopsy, periarteritis of the artery, periarteritis causing nodules, and periarteritis nodosa of the right upper quadrant will cause avascular or also called acalculous cholecystitis, and they keep asking this question, so you have to know all that. What is on the slide, we already just did. These peoples’ prognosis is very poor because of vasculitis. They should have surgery right away because they get gangrene very fast in the gallbladder.

So based on that, Question No. 2, same thing. You see the patient had Hepatitis B in the past, vasculitis, right upper quadrant, and leukocytosis in a 72-year-old, no sludge in the gallbladder. There is no stone, but there is sludge, or no sludge, either, edema of the gallbladder, so the next step will be a HIDA scan, which is D. In Question No. 3, bullous lesions on the hand, so this would be Hepatitis C or B. This is porphyria cutanea tarda. That means pigmentation, bullous lesion, and hypertrichosis. That means Hepatitis C. This question is always on the exam. It was asked in 2008, last year. The patient had Hepatitis C, comes with photosensitivity of the knuckles in 2008. Photosensitivity of the knuckles is due to porphyria, so you look for the word ‘photosensitivity’ with Hepatitis C because you will be asked about porphyria.

Question No. 4, on the other hand, has purpuric lesions like this. Serum complement is low; rheumatoid factor is very high, cryoglobulinemia. Did you know that rheumatoid factor is
The frequency of cryoglobulinemia is higher than in rheumatoid arthritis itself. In other words, when you see rheumatoid factor positive, you normally would think of rheumatoid arthritis. No. Think of cryoglobulinemia and cryoglobulinemia occurs either following Hepatitis B or C. This is also cryoglobulinemia, B or C.

All these questions are there when you go, and No. 5 is decompensated liver disease.

**Minimal indirect hyperbilirubinemia in a young man.** A 14-year-old male presents with fever, malaise, mild cough with upper respiratory infection and anorexia. Total bili is 2.3, while the indirect is 1.6, minimally elevated. AST, ALT, and alkaline phosphatase are normal. What is the next step?

If you see a young person or a boy in the exam with very minimal indirect high bilirubin, you're talking about Gilbert Syndrome. Any stress, stress can be a cold in the throat, can be a surgery, could be appendectomy, whatever stress of the child, if he has Gilbert Syndrome, which meaning by he has deficiency of UDP transferase, whatever that is, in the liver, that he cannot conjugate bilirubin, so you get slightly elevated, very, barely slightly elevated indirect bilirubin in the blood, only due to stress. Now unfortunately when you go to the exam, they don't give you the level of indirect bilirubin. They give you total and they give you direct, and you will have to subtract the two without a calculator. [Laughter] All right? And if it's very, very slightly high, you may think nothing of it, but that would indicate Gilbert Syndrome. Even a little fasting will make it go up, so all you do is repeat the test after the stress if over. It will be normal, so that's the whole question. Next?

**A 34-year-old female went on vacation to India.** On her return, she has jaundice, fatigue, and enlarged liver. There's no history of alcoholism. Her lab work shows AST 659, ALT 908, alk phos 104, bilirubin 5.5, serum protein of 11, albumin 3.4. Serology for Hepatitis A, B, and C is negative. ANA is 1:320. Diagnosis?

Autoimmune. We did that before. Whenever you see serum protein that high, 11 grams, wow, ANA positive, coming from India, that's autoimmune. Now when you see in your exam liver dysfunction, the first thing you have to decide is is it hepatocellular or cholestatic? You will see [inaudible] alkaline phosphatase. Is it mildly elevated? Then you know it is hepatocellular. If it is very much elevated, it is cholestatic. It is important to differentiate the two because the causes are different. This will be autoimmune based at hepatocellular. Primary biliary cirrhosis is cholestatic. Hemochromatosis is hepatocellular. PSC, primary sclerosing cholangitis, is cholestatic. Wilson's disease is hepatocellular, and of course cancer is hepatocellular, and each one has a different test, so mainly if the alkaline phosphatase is very much elevated, you would think of doing anti-mitochondrial antibody or ERCP, which is cholangiography. Otherwise you will do hepatocellular test. In autoimmune like in the case we just did, ANA and anti-smooth muscle antibodies are positive, the treatment of that condition is steroids. Autoimmune is treated with steroids. So don't forget vacation in India, protein of 11, autoimmune. Okay, this is the next one. You're done with this? Anybody? No? Okay.

[inaudible].

Which one? This one?

Yes.

Okay. From 2:00 to 3:30 is the hardest time in a lecture room because this is the time you wish your seats will become beds. [Laughter] It's the hardest time for me, the hardest time for everybody. I see this every year, the same thing, so you have to go through with this. If you want to snore, that's okay. [Laughter] Are we done with this slide? Everybody's become slow. [Laughter]
Now this may shock you up a little bit. It is time to seduce her [Laughter] with all this handsome hair on my mind, nothing there, [Laughter] and just a guess. How many glasses of alcohol he's got in front of him compared to her?

[Inaudible].

Yeah. Every woman should watch that. This is the idea of dating. Dating doesn't mean 'dating' dating. Dating means time to figure out if the other person is alcoholic or not [Laughter] because if he's alcoholic, and I have one or two in our own family, they beat the hell out of a women after getting married, so it's not a laughing matter at all, so you must—all of you who are not married, make sure you marry a man, find out if he drinks plenty of alcohol because they will hide it and you will never know. They will hide it to the end. They are very smart. You should do their uric acid. [Laughter] Uric acid is high in all alcoholics. [Laughter] Do you know that? Board question. [Laughter] AST. How else you would you do? The AST and ALT, Board question. If their AST-ALT is less than 500, actually less than 300, 300-500, and AST is double than ALT, ladies and gentleman, this man is alcoholic, so you do that, you tell your boyfriend you will get married, you don't care if they are VDRL or HIV negative or positive. [Laughter] You want to know the AST and ALT and do their ratio, 2:1. If the ration of AST to ALT is more than 2:1 and AST-ALT is less than 500 or 300, you know he is drinking. You are not going to get married. I don't care how much in love you are with him. Love will disappear a year after the honeymoon. [Laughter] After that it will be money-moon. [Laughter] For these people you have to be very, very—this is a Board question. I was just trying to dramatize to you to tell you that this is there. They will ask you how do you make a diagnosis of alcoholic liver disease? That's how we do it.

No. 2, end-stage liver cirrhosis due to alcoholism in a male patient. Presently he is not drinking alcohol. What do you want to do?

[Inaudible].

Liver transplant. If he is not drinking, if you're sure, you give him another liver so he can drink more. [Laughter] All the people in Hollywood, they're all alcoholic, they all get liver transplant very easy because they pay. The people who need liver transplants are not getting it. They get it. Then there is a condition called diabetic liver disease, which is going to be the future of the country. Everybody is eating too much food, everybody has become insulin resistant, and everybody is getting diabetic liver disease, called NASH. It's called non-alcoholic steatohepatitis. Prevalence—this slide was made two years ago. At that time prevalence was 24%. I guarantee you today prevalence of fatty liver in the U.S.A., 30%, and 20% of them will get liver cirrhosis. Imagine! People are eating all the time. We are eating while walking, eating while driving, eating in the restaurant, outside the restaurant. Too much food and triglycerides are high. Okay? This is there, in the exam. So if you go to the exam and you find liver dysfunction with diabetes, there are two conditions. They are both in the exam. One is NASH. The other is hemochromatosis. In the case of hemochromatosis, they have erection problems, too. What do they call that nowadays on the TV?

So the next one on the next page is diabetic, arthritic, liver dysfunction and loss of libido.

**Issue with diabetes, arthritic, and liver dysfunctions comes with loss of libido.**

So you’ve got hemochromatosis. Loss of libido is due to involvement of the pituitary gland. Well, if you have arthritis, you really don't need libido after that. [Laughter] And you have one minute for three questions.

[Three questions answered]
On Question No. 1, the gradient is how much? 3.0 minus 2.3. The gradient is less than 1.1, so if the gradient is less than 1, the conditions would be in your notes and also on the slide. Therefore, based on that, the answer is A. If the gradient was more than 1.1, then you’re thinking of liver cirrhosis or Budd-Chiari syndrome, so that’s the kind of question on the exam. There’s only one question like that, but it is there where you have to check the gradient and go by the causes. Unfortunately, you have to remember all those.

Question No. 2. Two is always on the exam, always, for a few years now. It’s called hepatic vein thrombosis due to the estrogens, so if you have estrogen plus ascites, you’re thinking of hepatic vein thrombosis, which is also called Budd-Chiari syndrome. For which you do Doppler ultrasound of the hepatic vein, so the answer is A. All these questions are there, okay?

No. 3. Three is a patient who has liver cirrhosis and presents with a new onset of ascites. I really don’t care if a patient with known cirrhosis has any change in anything. To you he could be talking different, he could be confused, he could have a little temperature. Any change, the textbook says, any change in the patient with liver cirrhosis, you should immediately think of spontaneous bacterial peritonitis. Of course, the ascites has to be there, and if you’re thinking of spontaneous bacterial peritonitis, you’ll do immediate ascitic tap and you take it to the lab yourself because you cannot leave the ascitic tap lying at the nursing station forever. You take it yourself to the lab and tell them to count the neutrophils or WBCs. If more than 500 WBC, the diagnosis is made. This is usually due to E. coli, and therefore you always give them Claforan for five days intravenously followed by prophylaxis with norfloxacin forever. These people tend to die from this condition, and therefore the Board will test it. All right? Next.

Question 1. The patient has been drinking alcohol heavily and comes to the E.R. in a coma. His friend tells you that the patient has been on over-the-counter medications for back pain. AST is 11-500 units.

11,500 units.

Sorry, sorry. [Laughter]

11 on this side and 500 on that side. That’s not fair to you. [Laughter]

ALT is 9,600 units, PT is elevated. What is the diagnosis?

[Inaudible].

Yeah, whenever the liver enzymes are in the thousands, you’re thinking of fulminant hepatic failure, and it’s usually in the exam from Tylenol. Even if you take a small amount of Tylenol, if you are alcoholic you’re going to get this. Only 3 to 4 grams of Tylenol can cause this condition, and it will occur very, very fast, within eight weeks. How will you know? You’ll be confused or your prothrombin time will be high. Your time on this earth will be less, but your prothrombin time will be high. [Laughter] Something will be prolonged. So once again, minimal elevation of liver enzyme in the exam for you means either Hepatitis C or fatty liver. They could even be normal. Around 300, I told you think of alcoholic liver disease. Between 300 and 3,000, viral hepatitis of any kind. Over 3,000, fulminant hepatitis either due to Tylenol or ischemia. Ischemic liver disease that goes into congestive heart failure, goes away the very next day. The patient comes with 6,000 AST. I treat the congestive heart failure, the next day is normal, liver enzymes are normal. I have seen that countless number of times, so that’s also fulminant hepatitis. Out of these causes of fulminant hepatitis, the middle three, the ones in yellow, have been on the exam. Acute fatty liver of pregnancy, we will do it in Ob-Gyn when we do that.
Day One

No. 2?

Which of the following is the absolute contraindication to liver transplant?

Active alcohol abuse. I showed you Tylenol toxicity when the glutathione is used up. That's why I got confused, but the liver transplantation indications are on the slide, and the pH is less than 7.3 and prothrombin time is over 100, and the contraindications are active alcohol abuse, so they will give you a patient with Tylenol toxicity and they will tell you the pH is 7.2. What are you going to do? The answer is transfer the patient to an institution where they do liver transplantation, or they'll give you prothrombin time over 100. The contraindication is alcohol use, active. There are many others, but I don't want you to know those because they're not in the exam.

No. 3?

A young patient is jaundiced and signs of psychosis. What is your next choice?

They've got hemolytic anemia, Wilson's. Okay. No. 4.

Seventy-three-year-old alcoholic patient with a past history of blood transfusion has acute CHF. Blood pressure is persistently low. Liver function test shows AST and ALT in many thousands and alkaline phosphatase almost normal. Total bilirubin is much elevated. Hepatitis B surface antigen is negative. The cause of abnormal liver function is?

Ischemic hepatitis.

No. 5?

A 34-year-old patient is brought to the E.R. because of confusion related to Tylenol toxicity. Which of the following is the best prognostic parameter?

For acute disease it's prothrombin time, like this one, but for chronic it is serum bilirubin or albumin.

Question No. 6. A 52-year-old patient with chronic liver disease due to active alcoholism is presented with decompensation, PT of 17 with an INR of 3. He is bleeding actively from esophageal varices. Patient is getting blood transfusion, FFP, Vitamin K, and octreotide. Endoscopic therapy fails to control the bleeding. What is the best mode of management at this time?

The answer is TIPS. TIPS is used to treat consequences of portal hypertension. It's a useful temporizing measure in those awaiting liver transplantation, and once you do TIPS, they get encephalopathy in 35% of the cases, so bleeding is controlled but they get confused. So you have two questions in two minutes.

[Two questions answered]

So In Question No. 1 you have palpable purpura and arthralgia, skin biopsy showing vasculitis, and rheumatoid factor is high, cryoglobulins, but this is cryoglobulinemia due to Hepatitis C.

Question No. 2, the answer is C because the patient has autoimmune hepatitis because of the high anti-smooth muscle antibody. All right? I know it's getting very boring at this time and you would like to go home, but you cannot. [Laughter] I'd like to go home, too, but now you do four questions in four minutes. Keep working. Work hard. Do fast.
[Four questions answered in four minutes]

Well, in No. 1 the alkaline phosphatase is very high, so that can only go with B because A and C go with autoimmune hepatitis, which is hepatocellular and antimysial is celiac disease, antimysial antibody is done for celiac disease, so the answer has to be antimitochondrial antibody for PBC, primary biliary cirrhosis. Primary biliary cirrhosis will give you from antimitochondrial antibody where the alkaline phosphatase is very high, and this is a patient with primary biliary cirrhosis. They have xanthomas, they have other autoimmune disorders like thyroiditis, Sicca syndrome, CREST syndrome, Raynaud’s, and they have high bilirubin, high alkaline phosphatase, and high antimitochondrial antibody, AMA. There’s always one question on primary biliary cirrhosis on the exam. These patients have pruritus, pigmentation, xanthelasmas, thyroiditis, osteoporosis, and everything else, a middle-aged woman, usually. Here are the xanthomas, the tests are non-specific except for antimitochondrial antibody, which in high titer is quite specific. They have high cholesterol, IgM is high. So the treatment of this condition for pruritus would be you give them cholestyramine or rifampin. They have vitamin deficiencies, fat soluble vitamins, and until you give them definitive treatment, you give them also deoxycholic acid. Okay? Can I move? Otherwise I will go to sleep. [Laughter]

Okay, Question No. 2 is about—what is Question No. 2 about? [Laughter] Biliary cirrhosis, primary biliary cirrhosis, and the best therapy is liver transplantation, B, like in ‘boy.’ No. 3, the patient is vomiting with right upper quadrant pain. Ultrasound shows gallstones. What is the bile duct size?

Normal. [in unison]

Normal. What is a normal size? Up to 6mm. If somebody had their gallbladder out, then the bile duct can also go up to 8mm as normal, but if the gallbladder is not removed then the normal bile duct maximum is 6mm. This is 4mm. That means there is no disease in the bile duct, and patient has been vomiting and right upper quadrant pain, and ultrasound is showing gallstones, but that means there is gallbladder disease, so you should do HIDA scan. HIDA should be positive because the gallbladder will not show up on the HIDA scan; therefore the answer will be acute cholecystitis, so the answer is HIDA scan because high def scan is done for cystic duct obstruction.

[Inaudible] ERCP?

No. ERCP is done for the bile duct. Bile duct is 4mm, which is normal. That means no need for ERCP. Because the HIDA is positive—if it is positive in this case, that indicates cystic duct obstruction, which means the gallbladder is not functioning, and the gallbladder is not functioning because of the gallstones.

No. 4, going for hip surgery, right upper quadrant pain, dilated gallbladder, and thick wall of the gallbladder. HIDA scan fails to show the gallbladder, so that’s again, acalculous cholecystitis; therefore cholecystectomy is the answer, cholecystectomy. This is an acute gallbladder because the HIDA is positive. That means cystic duct is obstructed. That means cholecystectomy. You see problem with this question?

[Inaudible].

You want to give antibiotics? For what?

[Inaudible].
The cause of acalculous cholecystitis is always ischemia. If you remember, I told you ischemic cholecystitis occurs in old people with arteriosclerosis, or it can occur from positive Hepatitis B antigen and that Hepatitis B antibody will cause periarteritis nodosa, which is vasculitis as a complication, which is also ischemia, vasculitis, so noncalculous, acalculous cholecystitis is always due to ischemia, so why antibiotics? So the secret is when somebody comes to you with right upper quadrant pain and the liver functions are normal or about normal, you're thinking of gallbladder, the next step is to do ultrasound because ultrasound shows the size of the bile duct. You must write that on the prescription. I write down ‘ultrasound for the size of the bile duct’ because I'm only interested in—you may be interested in the stones in the gallbladder. I'm not that much interested. I'm more interested in the size of the bile duct because my problem is if the bile duct is dilated, we have to do ERCP and the infection is in the—the stone is in the bile duct. If there is no obstruction in the bile duct, that means the bile duct will be 6mm or less. Then I'm not worried about the bile duct or the ERCP. Then I worry about the gallbladder, itself. The gallbladder, itself, means cystic duct obstruction, which are due to gallstones, so I do an ultrasound. It will tell me, well, the bile duct is normal but the gallbladder shows—doesn't even show stones. It shows sludge, and the amylase and lipase is elevated, so I know it's pancreatitis due to the sludge, so—or if there's no pancreatitis, it's just sludge and then I do the HIDA, the HIDA shows cystic duct obstruction, meaning HIDA is positive, then I go for cholecystectomy.

Everybody has it now clear? Those are the kind of questions they will ask you, speaking about the bile duct, and the gallbladder is diseased due to the gallstones. On the other hand, pancreatitis can come from gallstones or pancreatitis can come from—which we'll do right now, high triglycerides. We're going to do that. So if you can do the next 11 questions in six minutes, 13 questions in six minutes. I know it is tough, but you have to do it because I'm already Board certified. I don't need all this. [Laughter] I really don't need it. I don't care. Even if I know wrong, I know wrong. You have to file this exam, and each of these questions are going to be there, and then it seems to me they're not showing or teaching you at the universities, wherever you do your residency. They're not talking specifically about these things.

[Thirteen questions in six minutes]

So Question No. 1 has X-ray showing calcified gallbladder, which is like this or this, either this or this. When they tell you about calcified gallbladder on the exam, it is a pre-cancerous condition. Symptoms or no symptoms, cholecystectomy should be done.

No. 2 is emphysematous gallbladder, which looks like this. Gas in the lumen and within the wall of the gallbladder. Mortality is very high. Therefore you need to do cholecystectomy. There's an ischemic process causing it. This is also due to ischemia and also due to a gas-forming organism such as Clostridium [inaudible] or Clostridium perfringens, which causes infection and then ischemia, so it may have calculous, it may not have calculous. Again, surgery is indicated.

In Question No. 3, which is always, always, always on the exam, is a patient who had a hemicolecetomy in the past for ulcerative colitis. Any patient in the exam with a history of ulcerative colitis can develop—we call it PSC, primary sclerosing cholangitis. PSC occurs in patients with ulcerative colitis—only in the exam, [Laughter] and they have high alkaline phosphatase, and if you do the ERCP, obviously the ultrasound will show dilated, all strictures bile duct, and ERCP will give you the strictures like this. In primary sclerosing cholangitis, you have to do papillotomy, dilatation of the strictures and stenting, eventually liver transplantation, so in this patient, Question No. 3, the answer is ERCP so that you can make this diagnosis. At the same time do papillotomy and stenting until you find a liver later on. This question with ulcerative colitis is always on the exam. How will you know? The patient with history of ulcerative colitis will have high alkaline phosphatase. That's how you know.
No. 4. Look at No. 4. The bile duct's diameter is 1cm. It is cm. We were telling you normally its 6mm. 1cm means 10mm. 10mm means extremely big bile duct like this one. See the size of this? The size of the scope itself. This is the scope and this is the bile duct with two stones in it, so bile duct is big, so therefore you do ERCP and you find these two stones. When you find these two stones, you have to take them out by doing papillotomy, so the answer is B like in 'boy.' How do you do papillotomy? You put the sphincterotome inside and apply current and make it—cut the bile ampulla like that, and as you cut the ampulla, the stone will then come down. If it doesn't come down—this is the stone—then you put a balloon right above the stone, deflate the balloon, bring it up here, inflate the balloon, then pull the balloon out, and you will see the stone coming out into the ampulla, so this is—the answer is B like in 'boy' because of the dilated bile duct.

No. 5. Patient has pancreatitis but the HIDA is negative. If HIDA is negative, that means the gallbladder is functioning. The cystic duct is open, but there's sludge in the gallbladder. You know that the pancreatitis is due to the sludge in the gallbladder, and therefore the answer would be biliary microlithiasis. Biliary microlithiasis is a cause of pancreatitis. These are the causes of pancreatitis, and you need to look at it. There are only two main causes, alcohol and gallbladder disease. Others are very rare. Out of those rare ones, hyperlipidemia, you are expected to know, from the Board's point of view, and hyperlipidemia means high triglyceride level, so when I see a patient with pancreatitis, all I do is order an ultrasound of the gallbladder. I take the history of alcoholism and then do a serum triglyceride level and I will always have the cause, so to Question No. 5 the answer is A.

For Question No. 6, severe epigastric pain, denies alcohol use, amylase is high, so that's pancreatitis, so obviously you have to do ultrasound. The patient does not drink alcohol. Ultrasound is the answer, D like in 'doctor.'

No. 7, amylase is normal, triglyceride is 2,000. If the triglycerides are 2,000, in the lab, high triglyceride will interfere with measurements of the amylase, so even if the amylase was high, it will look normal because of high triglycerides. That's why people who have pancreatitis due to high triglycerides will have normal amylase.

[Inaudible].

Yes, lipase is affected to some degree. Lipase is more specific, but that is why on the Board exam they won't bring lipase into the picture. [Laughter] They will tell you patient has acute pain in epigastrium going to the back, amylase is normal, lipase is over 1,000. It has to be over 1,000 to do this problem, and they will then give you a tube of the blood of the patient, overnight stay in the refrigerator, and in the morning the two upper parts are milky, which is nothing but triglycerides and chylomicrons, and the skin of the patient is showing you the xanthomas of triglycerides, which is due to lipoprotein lipase deficiency, so the point is that in this case you have to dilute the serum and repeat the amylase. Then the amylase will be elevated, only if you dilute the serum, so if you have a classical pain case of pancreatitis in the exam with normal amylase with high triglyceride, you know the cause of pancreatitis is fiber glomerular [phonetic] amylase, is hyperlipidemia or hypertriglyceridemia. That's what they want from you.

The other thing in the exam is No. 8. In No. 8, here is the opposite of the previous one. The patient has no pain. He had pancreatitis. He is much better. He comes asymptomatic, goes home. Repeat amylase is elevated. Now amylase is elevated and patient has no symptoms. Opposite of the previous one. That means patient has pancreatic pseudocyst. In pancreatic pseudocyst the amylase will be high, the patient will be asymptomatic, opposite of the previous one, so you do a CAT scan or ultrasound. You will see the cyst. With a cyst you don't do anything until it gradually increases in size, so you watch it, observation.

[Inaudible].
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Pardon?
The answer to Question No. 8 is B. No. 9 is patient has acute pancreatitis. Five days later he is much better, free of pain, tolerating food. Amylase is high, but there is a cyst, a pseudocyst. The next step is continue the current medical treatment because this is an asymptomatic pseudocyst, which should only be observed. Nothing to be done unless it increases in size. If it was symptomatic then you will do ERCP and drainage or whatever you want to do, but if it is not changing or if it is not giving any pain, you don’t do anything, either. Just observe.

No. 10, the answer is A because this patient has developed acute ileus and leukocytosis after acute pancreatitis, and that is due to pancreatic abscess. This question is always on the exam. You have to do aspiration of the abscess or the pus of pancreatic abscess. A is the answer. This is a chronic question, always on the exam. The patient is very sick.

No. 11, we did before. It’s a calcification in the pancreas and the patient has abdominal pain. If you have chronic pancreatitis with chronic abdominal pain and morphine and Demerol don’t help, you’re supposed to give them pancreatic enzymes because if you give the enzymes from the outside, hopefully the pancreas says, okay, I will not produce my enzyme because it is coming from outside, and it is the pancreatic natural enzymes which dissolve the pancreas and cause the pain, so hopefully if you give outside—you do give enzyme, hopefully pancreas will produce less enzyme and the pain will be less. Sometimes abdominal pain of chronic pancreatitis goes away when you give pancreatic enzymes from the outside with each food. Each time they eat food, you give them pancreatic enzymes.

No. 12, there’s a malignant mass in the head of the pancreas, so that’s more chronic pancreatitis with pancreatic enzymes, and you always give pancreatic enzymes with H2 blocker because otherwise they are destroyed by the stomach. Acid destroys pancreatic enzymes, so always give H2 blocker when you give pancreatic enzymes. In pancreatic cancer, with cancer of the head of the pancreas like this one, now therefore the pancreatic duct is constricted, is obstructed, so you have dilatation of the pancreatic duct. Bile duct is constricted due to dilatation of the bile duct, so you have a double duct sign in cancer of the head of the pancreas. A double duct sign is like this. See the two ducts, the pancreatic duct dilated and the bile duct dilated, and that is the scope, so you have a double duct sign of the cancer at the head of the pancreas. When you see that, that’s your diagnosis. In this case, No. 12, there’s a malignant mass but no metastasis, so it is a localized cancer, therefore this should respond to Whipple’s procedure. Whipple’s procedure is a surgery done for pancreatic cancer if there’s no metastasis.

In 13, in the case of the patient with cancer of the pancreas, double duct sign, it’s all over with widespread metastases. If there’s metastases, there’s nothing you can do but maybe hopefully put an endoscopy and a stent to decrease the jaundice to some degree, make him comfortable so that he can sign all the money he has to you before he dies. [Laughter] There is nothing much that can be done about this except to put a stent, and this question is always on the exam. We put a stent in a case with metastasis, but if he does not have a metastasis, Whipple’s procedure, okay? Those are the two questions on the exam.

[Inaudible].

Pardon?

[Inaudible].

No. 13, the answer is B like in ‘boy,’ ERCP for stent, and like this is the stent. We are putting the stent here. This is the cancer all over. You’re putting the stent through it so that you can
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drain the bile to some degree, so you increase his life by a few more months. This is a biliary stent, in the duodenum draining it, so it just comes out by itself after awhile, and that’s the end of the story, usually.

So let’s go to the New Questions.

A patient with a history of Hepatitis C and liver cirrhosis presents with nephrotic syndrome with significant protein greater than 5g in 24 hours, hypo-albuminuria. Her only medications are Lasix and Aldactone daily. What is the diagnosis?

This has never been asked, this question. What they’re trying to do here; looks to me, that glomerular disease may occur in patient with chronic HCV infection. That would be new. In other words, a complication of Hepatitis C is glomerular disease, and it is usually membranoproliferative glomerulonephritis, usually in some people associated with essential mixed cryoglobulinemia. As you know, Hepatitis C can cause cryoglobulinemia, and cryoglobulinemia is associated with membranoproliferative glomerulonephritis, and I’ll show you some slides on that when we do kidney disease. Less frequently you can get membranous nephropathy. Several series have reported that Hepatitis C antibodies are nearly universal in patients with both membranoproliferative disease and cryoglobulinemia. The pathogenesis appears to relate to deposition of immune complexes containing anti-HCV and HCV RNA in the glomeruli, so it looks like the antibodies to Hepatitis C go and hurt the kidneys, too, causing glomerulonephritis.

May I have one, too? We are all having gum? [Laughter] What’s wrong with the teeth?

[Inaudible].

Oh, you keep yourself awake with gum? [Laughter] Gum in my language means very unhappy. [Laughter] In your language it’s ‘gum here,’ and my gum is really unhappy. [Laughter] So you need gum. [Laughter]

Okay, so in other words, what they tried to do last year for the first time, in a patient with Hepatitis C who gets nephrotic syndrome, its membranoproliferative glomerulonephritis due to Hepatitis C.

Question No. 2.

Which of the following tumors of the liver is caused by vinyl chloride?

Vinyl chloride causes angiosarcoma in the liver. Angiosarcoma of the liver occurs with vinyl chloride, and I could not find this in Up-To-Date. I knew a long time ago that vinyl chloride also causes scleroderma-like picture. Actually, not only angiosarcoma, these people get sclerosis of the skin and scleroderma-like picture, and I knew that a long time ago, but I couldn’t find the references for this, so you check it. My impression is the answer is angiosarcoma.

[Inaudible].

What?

[Inaudible].

Okay. I don’t understand. The echo is difficult for me, but whatever you say is okay. [Laughter] Oh, you said Robin’s hepatology book. First of all, I didn’t know the hepatology book by Robin. [Laughter] Secondly, I trust your judgment, and, number three, I knew about it a long time ago, and, number four, its
good now, everybody else knows about it. [Laughter] But this was never asked before. It's the first time, so I don't know if it will be asked again or not. It probably was experimental.

And No. 3.

A 22-year-old female comes with abnormal LFTs, fatigue, diarrhea, and some jaundice. Some of her coworkers were sick like her. You treat her symptoms and she comes back in two weeks for follow-up. There is now no diarrhea, lymphadenopathy, jaundice, or hepatomegaly. LFTs are normal. She still has some fatigue. What is your next step?

I really couldn't understand this question much, but I'm thinking check for Hepatitis A, at least. It's a young girl. Hepatitis A can come to a young person and can go to another person, so I would at least check for Hepatitis A. You take whatever you want. I just don't understand. See, the thing is this. When people send me the questions, there is a good part and a bad part. The good part is that you know exactly now what they're asking, all right? But the bad part is you cannot blame them sending me a question after the exam is over and they've sent me 50 questions, so they're all hodgepodge, mixed in the blend, so one question goes into another, so I get this mixed information. Then I sit down and try to figure out exactly what they're trying to ask, so if there is something like this in the exam then I am putting my bet on Hepatitis A, but obviously I could be wrong and there could be something else in the question which is missing here, so I'm just giving you as it is. I'm trying to be as honest as I can to give you what I am given, okay? I can only give you what I am given, and this is the way it is. Whatever it is, it is, okay?

No. 4?

Which of the following carries an increased risk for the transmission of Hepatitis C from a husband to his wife?

[Inaudible].

Sharing toothbrush? [Laughter] Well, that's the height of poverty [Laughter] Out of these four, I'm taking blood transfusion. That is the most common cause, blood transfusion or IV drug use. They didn't mention that. Sexually you can transfer it, but very rarely. It happens, but not that commonly. Toothbrush? I don't think they should share it. [Laughter] And kissing, I don't think you can spread Hepatitis C that way, unless you are kissing like— [Laughter] I don't know what to tell you.

On Page 37, I give you some timesavers, which we have already done as we went, especially to remember the bloody diarrhea in Milwaukee, and just remember—read the rest of them, cobblestone and prostitution. You probably already know all these now, but that's one way of going over just the day before the exam.

Sometimes these images—all these images have been asked before in the exam. Not that they're in every exam. They just scatter them around. Because I understand now they give three or four booklets. In other words, everybody doesn't get the same exam, right? They make three different books in [inaudible] and they give it to you. It could be the same books but mixed, Question 1 for 31 or something like that, or it could be a different book, similar, but the standard remains the same. For example, on the same disease one will be asked the diagnosis and the second one just the treatment, so it just means the same thing. And then I give you—at the end of each subject, I give you my impression of questions. The thing is this. People come to me, you mean to say this is what you give us, then I'm going to do this very well and you're going to pass the Board? I mean, I'm already done with it. I'm already done with your material. Now what do you want me to do? [Laughter] I get defensive. I say, all right, what you do is do you have MKSAP? Oh, yes, I've got MKSAP. Okay, you just do—did you do MKSAP at the
end of chapter? There are a total of 100 questions, around 100. If you did all of those, it's a waste of your time. I went all over them, and I picked up the questions which I thought were the standard of the Board, but most of the questions in MKSAP are not the standard of the Board. Actually, they're higher standard. We don't need that. You only need a few out of these, okay? [Laughter]

I found out there were one-third of those questions which were good for you, so in order to save two-thirds of your time doing all the questions on MKSAP, only for those people—believe me. Those who have done my material three to five times, only after that if you want to practice, add to your knowledge, then I want you to do these MKSAP questions. For example, in GI I picked up 50 out of 142 questions. Saves you so much time. Instead of doing 142 questions and wasting time, plus you will get confused and your brain will be going left to right, so just do these ones, 50, and that is just for practice because it's only after you have done my material so that you don't call me and complain that you want something else to do. All right? So we will now break for—you want to say something? We will break for a cup of coffee. Come back in 20 minutes and we'll start all over again with Nutrition. Thank you.

**NUTRITION**

There are about six questions or so, or seven to eight questions on the exam, usually. One is atrophic tongue, which is iron deficiency. Iron deficiency causes atrophic tongue. That means loss of papillae, like this one, loss of papillae. It is iron deficiency. Now this is not a painful tongue. If the tongue was painful, which is this one, then they should give you high MCV, which is Vitamin B12 deficiency due to inflammation. Such patients will complain of paresthesia of the feet because of B12 deficiency. MCV is high. Fissured tongue is also seen in Vitamin B12 deficiency.

Then there's a question on pancytopenia. What is the name of the vitamin deficiency which causes pancytopenia? That is Vitamin B12 deficiency. B12 deficiency causes pancytopenia. B12 does a lot in the examination. B12 is their darling. [Laughter] Deficiency of which of the following vitamins increases the blood level of homocysteine? [Inaudible].

Both folic acid and B12, so there are Vitamin B12 deficiency related questions on the exam: number one, vegetarian, number two, pancytopenia, high MCV, high LDH. Megaloblastic madness. These people go crazy, very angry. For those women whose husband gets angry, they should do the B12 on them, also, [Laughter] and if they have spastic legs, then you should be able to tell. They have no vibrations left, and their methylmalonic acid and homocysteine levels are high, so these people are spastic. They get angry easily. On top of that, they are vegetarians. [Laughter] So Question No. 5, deficiency of which vitamin increases the blood level of homocysteine, as well as methylmalonic acid? Vitamin B12. Six questions, three minutes.

**[Six questions, three minutes]**

So, Question No.1, The answer is C because this is a description of subacute combined degeneration of the spinal cord that can occur without anemia. In other words, if you have B12 deficiency, and you get subacute combined degeneration, you don't have to have anemia. This patient is ataxic.

No. 2 is stocking-glove neuropathy in the legs. They give you Vitamin B12 of 220, which is normal, but it is in the low range of normal, so they want you to confirm the diagnosis.
Confirmation of the diagnosis is done with methylmalonic acid. Thank you very much. It's good to have the sound like this. [Laughter]

And No. 3, the patient is cake and cookies, depressed, confused, and there is one question on the exam where one patient has both vertical and horizontal nystagmus. I remember this question many years ago when I was failing a lot, and this is one of the reasons I failed this question because I'm sitting there forever thinking—I had remembered horizontal nystagmus means peripheral disease, [inaudible] disease [inaudible], or central disease with vertical nystagmus. That's how I remembered. I know this patient has both nystagmus. [Laughter] I'm thinking [Laughter] vertical and... Again, both diseases, peripheral and central? [Laughter] What could that be? Then I figured it out, finally. It is one condition on the exam. It is always there. It is thiamine deficiency and causes both vertical and horizontal nystagmus, so you will not now forget this, okay? Both, and the cakes and cookies go with that, so you get beriberi.

Beriberi can be wet and dry. It occurs in alcoholics, and in wet beriberi you get high output state, and in dry you get the nerve involvement. With the Wernicke you get nystagmus. You get Korsakoff syndrome. You write these answers first so I can go further? A, B, C, C, B, all right? In subacute combined degeneration, you get paresthesia, loss of vibration, spastic, ataxic, or [inaudible], all right? That's B12. They don't know where their joints are, loss of position sense. They don't know where their joints are or where their body is. You ask them where is your left leg? Some people will be driving and say, sitting next to me, go to the left, go to the left, left is that side, go to the left. [Laughter] In thiamine you get confusion, nystagmus, double vision, and postural hypotension, and sinus tachycardia. I will never forget this case in ICU. The patient was admitted and all these cardiologists, and blood pressure is low and won't come up, and they cannot figure it out, so they're giving a dose of dopamine to bring the pressure up, and I had just read this, so I went to the patient. The patient was alcoholic, so all I did was give the patient thiamine. The next day the blood pressure was normal without dopamine, so remember thiamine deficiency can cause hypotension in an alcoholic. Very important, and most cardiologists won't know about it. If you have tachycardia with hypotension, think of thiamine deficiency.

So in your notes, I have given you four under Question 3, four presentations of B1 deficiency in the exam: alcoholic patient with nystagmus; on the third post-op day, a retired college professor becomes confused and ataxic; and third is gastric bypass surgery, eats fast food in small amounts, he's now confused with eye disconjugation; and number four is after surgery patient has difficulty coming out of anesthesia, Wernicke's encephalopathy, so those are four scenarios of thiamine deficiency in the exam questions.

Question No. 4 is a classical description of pellagra. Pellagra is—this patient, necklace. See that? Dermatitis is in necklace form, so we call it four Ds, dermatitis, diarrhea, dementia, dermatitis, and death. That's nicotinic acid. This particular dermatitis on the exposed areas, necklace type dermatitis.

No. 5, lives alone, cakes and cookies and milk, and painful bruises like this one is Vitamin C deficiency. In the question in the exam, they give everything but bleeding gums. Bleeding gums is also seen in scurvy, called bachelor's scurvy, and in scurvy you get gums like this. How do you brush these teeth? [Laughter] They're so irregular. [Laughter] So talking about gums, what were these gums?

[Inaudible].

On the left side is Dilantin. On the right side is cyclosporin. Cyclosporin can also cause hypertrophy of the gums. Talking about gums, one more gums—gum. You're eating gum.
Blue gums. If you have blue gums and constipation, you’ve got lead toxicity—lead toxicity.

Before I go to it, Question No. 6, the answer is Vitamin K deficiency. The PT is elevated, and PTT is also. Both can be elevated with Vitamin K deficiency. Antibiotics destroy bacteria that synthesize Vitamin K. Those patients who are not eating leafy vegetables and are using antibiotics can get Vitamin K deficiency within one week and high PT and PTT.

Questions on mineral deficiencies.

Question No. 1, a 71-year-old hypertensive patient, while on captopril and chronic hyperalimentation develops dermatitis of the extremities and dysgeusia. What is the cause?

This is called zinc deficiency. In zinc deficiency you get dysgeusia. Diarrhea and dermatitis and loss of hair. You get acrodermatitis, loss of hair, loss of taste, and chronic diarrhea, but dysgeusia can also occur from captopril, medication you give to the patient, Capoten, can cause dysgeusia. This is zinc deficiency. This came in the New England Journal of Medicine, March 17, 2005. In a child born with zinc deficiency, acrodermatitis, zinc deficiency. I’ll never forget this case. Came to my office saying that—old lady. Only thing she said to me, and I still feel bad about it, that pain on the pulp of my fingers. I had no idea about this slide. I had no idea. I told her I don’t know what you’ve got, and she left, never came back to me. Later on I saw this slide. Pain on the pulp of the finger, you’ll now not miss, means zinc deficiency, these pains here, okay? So I missed this diagnosis in my office, I remember, but please you don’t miss it. If you see one, you can help one person. Amazing.

No. 2.

A patient on long-term TPN. Deficiency of which of the following minerals is associated with glucose intolerance?

[inaudible].

How many people know about this? Four or five? Chromium is the answer, chromium. Chromium deficiency can cause glucose intolerance, and chromium deficiency causes proximal muscle pain, so if you have your diabetic, you see, when they lose glucose in the urine, they lose chromium, also, and chromium deficiency causes glucose intolerance, so always think of chromium deficiency in your diabetic who is not controlled, and then he will start coming to you with neuropathy and muscle problems. Just think of chromium. Always chromium picolinate is available over-the-counter. Even a microgram a day for a few days takes away all the symptoms of neuropathy and myopathy. In general the nonspecific things they get. You don’t know what is wrong with them. You give them chromium. It doesn’t hurt, and diabetes tends to get controlled better with that.

No. 3, the patient is on long-term TPN. Deficiency of which of the following minerals is associated with cardiomyopathy?

[inaudible].

The answer here is selenium. Selenium deficiency causes cardiomyopathy.

On the toxicity questions, health-food faddist having headaches and dry skin and peripheral edema, calcium and mild liver dysfunctions are there. That’s a classical description of Vitamin A toxicity. They get headaches, high intracranial pressure from Vitamin A, dry skin, liver dysfunctions, hypercalcemia, and anorexia, bone pain, muscle pains.
Vitamin D toxicity also causes hypercalcemia, so the difference will be what? You notice this difference is dry skin because both can cause hypercalcemia. In Vitamin A toxicity you get
dry skin. All the other symptoms can occur in both, and they have that question on the exam, dry skin.

Okay, questions on enteral feeding.

A patient is five days status-post CVA, has recurrent aspirations. On exam there is saliva pooling into the back of his throat. Management?

That's papilledema.

Question No. 1; the answer—this was asked in 2008. Although most patients can be started on low-volume continuous intragastric feeds, a significant history of any of the following would favor starting with jejunal feeding. In other words, indications for jejunal feedings are written in your notes on the bottom. Those are the indications for jejunal feeding, but what is the answer to the question?

[Inaudible].

If the saliva is pooling into the back of the throat, I think you want a speech and swallow evaluation. I would take A. You want swallow evaluation. That is the management initially, and then I'm giving you the last three lines. I'm giving you the indications for J feeding. If you can prove that, then you can do your jejunal feeding.

Question No. 2.

A 62-year-old patient with acute pancreatitis is admitted to the ICU. He has been NPO for a few days and needs to be fed. Which of the following would be the best method of feeding during his acute illness?

If you read the underneath portion, the answer will be enteral feeding without lipids because this is acute pancreatitis. You often get ileus and abdominal pain, and the evidence suggests that enteral feeding is safe and may reduce complications. Enteral nutrition has the ability to maintain intestinal barrier. The American College of Gastroenterology and the American Gastroenterological Association recommend enteral feeding for severe acute pancreatitis, so whenever possible you should be doing that, so the answer is B like in ‘boy.’

New Questions.

A patient is hospitalized following a post-gastric bypass surgery. He remains NPO after four to five days and has had recurrent vomiting. He also complains of leg pains, especially on climbing up stairs. On exam he is somewhat confused and dehydrated. There is horizontal nystagmus. Diagnosis?

[Inaudible]

Horizontal nystagmus, B1 deficiency. Confused, horizontal nystagmus is B1 deficiency.

No. 2.

A 44-year-old man has gained 100 pounds of weight in the last few years in spite of following dietary and exercise recommendations. His BMI is 44. His thyroid and other hormonal work-up is negative. He has no contraindication to gastric bypass surgery. What would you advise now?

[Inaudible].
The indications for gastric bypass surgery are three, written underneath: BMI of more than 40—this one is 44, acceptable risk for surgery, and failed previous non-surgical weight loss, so therefore the answer is gastric bypass surgery.

**Question 3**, a patient with acute pancreatitis is in the ICU for ARDS and is status-post intubation. He has been NPO. What would be the best way to provide nutrition now that pancreatitis is healed, abdominal distension has decreased, and he is having normal bowel sounds?

Answer is same as before, nasojejunal tube feeding, jejunal feeding. The nasojejunal feeding was the answer in the exam, and that's because the enteral nutrients maintain the intestinal barrier, and meta-analysis of 27 controlled trials confirmed that enteral nutrition in the setting of acute pancreatitis was associated with significant reduction in length of stay and infectious complications, so therefore the answer was nasojejunal tube feeding, so that's something new that you should know in case they start to ask that.

[Inaudible].

Well, J-tube would be fine, too, but they didn't give that choice. So the time-savers are underneath for you, so we can now go to medical statistics.

**MEDICAL STATISTICS**

Let's do the medical statistics, which as far as I'm concerned is very difficult for me because I don't know anything about it. I never had any training in medical statistics. If there was a class, I never attended it. [Laughter] Usually with 8:00 a.m. classes, I never get up until 9:00 a.m., so all I know is I can make you pass this. The way I can do that is there are six questions on the exam on medical statistics. Three are on definitions and three are on calculations. I give you all those six questions and you pass, right? [Laughter] They don't need to know anything about it.

The first definition they want [inaudible] is P value. I have no idea what is P value, so I decided to write it down for you. It's all written for you. P value is a way of expressing a study's statistical significance. It determines whether or not an observed effect can be explained by chance. When P value is between 0.05 and 0.01, the result is usually called 'statistically significant.' When it is less than 0.01, the result is often called 'highly statistically significant.' That's easy. The lower it is, the better for you, all right? Big deal. The interpretation of P value can depend on design of the study, method of collecting data, and analytic practices used.

I tell you what. I have given you the last two pages in this document to really understand the meaning of P value. If you see the last two pages and that one, as well, it came in—it was very nice—Medscape Business of Medicine. "Michael Jordan Won't Accept the Null Hypothesis: Notes on Interpreting High P Values." If you read this, you will really understand the meaning of P value. My problem is I read it and I forget about it, my problem is I can't remember things, but you have to remember until the examination date. After that you can forget it, too. [Laughter]

The second definition on the exam, which is every year, is on meta-analysis, and meta-analysis means—it refers to the collection of methods for combining quantitative information from several sources to give a summary statistic. In other words, one professor gets up, decides to read, say, on acute pancreatitis, so he picks up 350 articles on acute pancreatitis and he decides which one is good, which one is not good, discards a few of them, sits down and writes his own stuff on acute pancreatitis talking about all these studies he read, so this is called meta-analysis, okay? So some of these people have nothing else to do, [Laughter] so they sit
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down and decide to do that. It’s called meta-analysis. If you fail to remember the definition of meta-analysis, I can tell you the answer on the exam is B like in ‘boy.’ [Laughter] Just in case on the meta-analysis question you don’t remember the definition, the answer on the exam is B, ‘boy,’ [Laughter] and you will pass.

Next is confidence interval. Now the question on the exam on confidence interval was, a medication is effective with a P value of 0.05 and a confidence interval of 95%. That is between -15% and +85%. What does it mean? I don’t know. [Laughter] It means that you are confident 95% of the time that there is an 85% chance that it will work favorably and a 15% chance that it will not work favorably, okay? That’s what it means. It’s no big thing. They’re just definitions, and you should remember the definitions, and that’s what they are. For example, on the next page, is the definition of randomized study. Everybody knows that. I don’t have to give you the definition of a randomized study.

On the determination of diagnostic tests, the question they ask is if you increase the threshold for what is normal, what will happen? You will get more negative results or more positive results? You will get more negative results, and this will decrease the sensitivity and increase the specificity. If you decrease the threshold for normal, you will get more positive results. For example, if you remember cholesterol, in my time the serum cholesterol normal was 250. In your time it is 200, so we decreased from 250 to 200, made it normal, less. What happened? More true positive tests, okay? So it’s the same thing. If you decrease the threshold for normal, you will get more positive results, and that will increase the sensitivity and decrease the specificity of the test. That’s what you’re supposed to know.

On the interpretation of the diagnostic tests, they keep asking the definition of sensitivity. Sensitivity is the ability of a test to correctly identify patients who have the disease. In other words, you know the patient has disease. The question is how sensitive is the test that can pick up that disease, so that’s a question, and you can calculate that by taking true positive and divide that by true positive plus false negative. That’s in your notes. Similarly you can calculate specificity by taking the true negative and divide that with true negative plus false positive. Unfortunately you have to remember these formulas in order to calculate the sensitivity or specificity if they ask it on the exam. The same way on Page 3. They will ask you how you calculate positive predictive value, and that is always on the exam. As a matter of fact, they will give you a question on the exam to really calculate the positive predictive value by giving you various numbers, which we’re going to do soon with you together. So you calculate the positive predictive value with a true positive divided by true positive plus false positive, and negative predictive value by taking true negative divided by true negative plus false negative. So now you have—based on this, let’s see if you get some idea of four questions in three minutes.

[Four questions in three minutes]

Very good, looks like you’re all done. Do they give you lectures on medical statistics at your universities? They have? That’s why you will definitely know better than I do because to me this is Greek really. For example, the formula for calculating sensitivity of a test, we gave you that in [inaudible], right? That is true positive divided by true positive plus false negative, and for negative predictive value, it is written above on the same page. And why do we do randomized studies?

[Inaudible].

How many people take B like in ‘boy’? That is the answer. And No. 4, how many people answered A? Okay, only a few are taking it, but I am taking A. My reason for taking A is this. Negative predictive value is the probability that a patient who has normal test is actually free of the disease, so lesser than normal test result, higher will be the negative predictive value, so
less of the normal test result occurs with the tests that are more sensitive. Therefore the higher sensitivity, the higher will be the negative predictive value, and therefore my answer is A, so I'm
going that route thinking that way, but you decide what your answer is. My answer is A, and this is a Board question.

On the next page, No. 4, somebody in the class last year gave me this. He says, doctor, this is a very easy way of remembering these, so I just put it there. I'm not sure if it will work for you. I mean, he puts the arrows and the boxes like that, and then he puts them by A, C, so A divided by A + C. To me, it's more confusing. [Laughter]

Let's go to the calculations on Page 5. Prevalence of a disease in a population is 10%, sensitivity of the test is 90%, specificity is 95%, and the question on the exam is what is the positive predictive value. Sometime in the exam they will give you like this 2 x 2 table, and they will even write exact numbers so you just have to calculate, but sometimes they won't give you the numbers and you have to find the numbers yourself, so let's do that together for a change just to see what happens. Those of you who are getting disheartened that this is very tough and cannot be done, I can assure you that you can do definitely right answers on the exam with the definitions. There are three total calculations, and I can also assure that one type of calculation I guarantee you that you will be able to do, which I'll come to you, but if they give you this, which is there on the exam, I'm positive they will ask you to calculate positive predictive value with these numbers, some of you will have difficulty, but it's okay. Out of six questions they give you, if you miss two it's still okay. I would rather you spend time on the other questions which are more important than this, okay? So don't get disheartened, but I have to go through this process, so that's why I'm doing it, otherwise I won't repeat it.

So let's do it together. Prevalence of the disease in the population is 10%, and always think that the population is at least 1,000 because then the calculation becomes easy. Ten percent of 1,000 is one hundred, so 100 have the disease, therefore out of 1,000, 900 do not have the disease, right? So you just remember that fact because you're going to need that 900 later on who do not have the disease. So let's go to the ones who have the disease. True positives are 90, okay, because sensitivity of the test is 90%, and therefore out of 100 who have the disease, 90% have the positive test. Therefore the true positive is 90. What will be the false negative? The rest of them, which is 10 out of 100, right? So 90 true positive, 10 true negative. So you put that on the 2 x 2 table, which I did for you, 90 on the top left, 10 on the left bottom. Now let's check the specificity. Specificity to the test is 95%. That means specificity by definition is those people who don't have the disease. That is out of 900 who do not have the disease, 95% have the negative test. Therefore true negatives are 95% of 900, which will be 855. That is 95 divided by 100 and multiplied by 900. Ninety-five percent of 900 will come to 855, so how many are false positive? Nine hundred do not have the disease, 855 are true negative, so if you subtract 855 from 900 you should get those people who are false positive, and that will be 45, and you put that on the 2 x 2 table, and now you calculate the PPV, which is true positive divided by true positive plus false positive. That is 90 divided 90 + 45, will be—if you calculate and multiple that by 100—always multiply that by 100 to get the percentage—it will be 66.7%, so the answer to the question is 66.7%, and you do it later on, but the details are written for you.

If you understand that, then you can do the next two questions, which is Question No. 1. Go ahead.

**PSA has a sensitivity of 75% and a specificity of 80%. Prevalence of prostatic carcinoma in your referral male population is 10%. If your patient has a positive result on the blood test, what is the chance that he has prostate cancer?**

That is to say they're asking what is the positive predictive value of the test? Again, I will do it with you. In the given question, PSA that is sensitive would be 75%, the disease prevalence is 10%, 100 out of 1,000 have the disease, but the sensitivity is 75%, so 75 out of 100 with the disease have a true positive test, and therefore 25 have a false negative test. The specificity of
the test is 80%. Of the 900 who do not have the disease, 80% have negative test, so 80% of 900 would be?

[Inaudible].

Seven hundred twenty. Perfect. Seven hundred and twenty are true negative, therefore 900 minus 720, 180 are false positive. Therefore the true positive is 75, false negative 25, true negative 720, false positive 180. You apply the formula of true positive divided by true positive plus false positive and multiply by 100, you will get 29%, so the answer is 29%. Similarly, if you do Question No. 2 later on, the answer is 12%. Twelve percent is the answer to Question No. 2, and, again, if you don’t know, if you get totally confused on the exam, just answer B like in ‘boy.’ Twelve percent.

I used to do that in the exam. In my time some of the questions would be true and false, okay? Each statement—there’s no best answer. They used to say, okay, A is true or false, B is true or false, C is true or false, D, true or false. I didn’t know anything about the questions. They will ask about the cellular level of the GI tract in GI subspecialty, so I didn’t know how to do it, so I thought, okay, if I had heard about these questions, I would have known that. If I would not have known it, I’m going to call all of them false. If I don’t know a question, the answer is false, okay? [Laughter] I knew if I did that, I’ll be 50% right. What I did, it either is true or it’s false. Fifty percent right, right? How can you lose? So very simple. Any question I encountered on the exam on true and false, I would say false. And guess what? I passed the exam. [Laughter] I didn't know the concept and I passed the exam because I took the percentage. I said, look, 50% are true, so after that, I wrote on the Board. They found out about that and they stopped giving true and false questions. [Laughter] No more true and false questions after that because]. So similarly, I said if you don’t know what it is, you will pick A, B, C, or D. Now the chances are much more meager to really do it, but, still, if you don’t know anything, decide which one you want to do, A, B, C, or D on all the questions you don’t know. You’ll still do some right.

Okay, now the other types of calculations on Page No. 7. This everybody can do this. This is easy. In this type of question they will give you two percentages, okay? When you see two percentages in a question, just subtract the two. For example, the mortality decreases from 6% to 3% on using a new thrombolytic drug in a disease. How many patients with the disease will you have to give this drug to save one life? In this type of question, you subtract the two percentages. Six minus 3 is? Three. You will be saving 3 lives out of 100, right? It’s a percentage. Three out of 100 means 33, answer is 33. You didn’t get it?

Go to No. 2. You’ll get it. You’ll see. No. 2, what will be your answer if the mortality decreased from 40% to 20%? Forty minus 20 is 20, so 20 out of 100, right? You will be saving 20 out of 100 lives. That means 1 out of 5. Five is the answer. Think about it.

Okay, go to Question No. 3. I’ll get there. Go ahead.

An HIV pregnant patient has 2% risk of transmitting infection to the baby if delivered with C-section compared to 7% risk if delivered vaginally. How many C-sections would you have to do to prevent one HIV infection?

Seven minus 2? Five percent. You will be benefitting 5 out of 100 people. That means 1 out of 20. Five out of 100 means 1 out of 20.

When the fraction is reduced.

So 20 is the answer.
So the answer is the denominator. Is that clear? Seven minus 2 is 5. Five over 100 reduces down to 1 over 20, so the answer is 20.

Try again, Question No. 4.

Three hundred subjects were randomized for a trial for the efficacy of a drug. The placebo group has 90% mortality at one year. The treatment group has 75% mortality at one year. How many patients need to be treated to save a life? Ninety minus 75?

Fifteen out of 100.

Over 100, reduces down to 1 in—

Six. Six patients need to be treated to save a life. This is easy, man. What's going on? [Laughter] Even I understand this one. [Laughter]

A patient with HIV has genital herpes. Risk for herpes to fetus with C-section is 2% and with vaginal delivery is 7%. How many deliveries do you have to do by C-section to save one fetus from acquiring the herpes?

[Inaudible].

Right.

20.

Seven minus 2, 5. Five over 100? One to 20, and 20 is the denominator.

All right, let them do No. 6. [Laughter] Who will give me the answer on No. 6?

Ten. [in unison]

Ten. Done. Easy. Okay, so there’re going to be two questions like that, and I’m sure you can do both of them. I’ll give you one more, which is easy. They will give you a graph like this on Page 8. I’m just talking about the top graph, okay? The rest is just explanation. In the top graph, which of the points indicates maximal sensitivity? The cutoff point is A and the cutoff point is B. Because there are very few tests that are simultaneously 100% sensitive and 100% specific, it is difficult to place the cutoff point to separate normal from abnormal. The test cutoff points will have effect on sensitivity and specificity, as shown here. Cutoff Point A is 100% sensitive, so that’s the way they put it. The cutoff point is 100% sensitive, therefore the answer is A. They’re asking you which of the points indicates maximal sensitivity. Cutoff Point A is the maximal sensitivity. That will identify all the individuals with the disease. Cutoff Point B, on the other hand, is 100% specific. Values below B will identify all normal individuals, and if Cutoff Point A is moved towards Point B to make it more specific, it will become less sensitive. As Cutoff Point B is moved towards Point A to make it more sensitive, it will become less specific, so the answer to the question is A. It doesn’t matter. If they give you a graph like this and the question is which cutoff point is maximum sensitivity, the answer is A, okay?

Now you go to the New Questions on the next page, No. 9.

Question 1. Drug A decreases serum cholesterol in 25% of patients. Drug B decreases serum cholesterol in 20% of patients. How many patients need to treat with Drug A to decrease the serum cholesterol in one patient?
Now be careful. This is not the similar question which we did before. Look what it is doing to you. They purposely did this. I was telling people to subtract the two percentages. Now that is not here. These are two different things. This is not the same type of question that you are using in a study.

Four. [in unison]

The answer is 4, C, 4 patients. Right. This is easy. It's different. It has nothing to do with the 20%, everything to do with 25% of the patients, which is 4 actually. It's so simple. No. 2, Question No. 2. What do you want to take as the answer to No. 2?

A 61-year-old patient is found to have a nodule in the right lung. A PET scan of the mediastinum shows an increased uptake. The thoracic surgeon is informed. He decided to do a mediastinoscopy. Why did the surgeon take the decision?

A, because of the sensitive test. Sensitivity is high, that's why, A. No. 3.

**What is the meaning that a test has a 67% sensitivity?**

It means only 67 patients out of 100 with the disease have the positive test. The rest have the disease but the test is negative, okay? Again, only 67 patients out of 100 with the disease have the positive test. The rest of them have the disease but the test is negative.

No. 4, and No. 4 is about Type I and Type II errors. They don't ask that that often. They did ask the last time as a new question, so I decided to put it underneath there for you to read about Type I and Type II errors, just the definition. And on Page No. 10, again, similar likelihood ratios and their definitions for you to study. Really, they have occasionally asked those, not always, but I still put the definition in the exam course for you to understand it.

No. 6.

**A study is done in a population of patients with high pretest probability of celiac disease. What would the study have to show for you to accept the validity of this study?**

Naturally, the lesser P value will be the answer. The answer is A. All right? So that is your medical statistics, about six questions on the exam. Don't worry about them.

**DERMATOLOGY**

Let's do dermatology now.

**Question No. 1, a patient with herpes labialis develops a target lesion. [Inaudible.] What is the possible diagnosis?**

Herpes labialis and the patient develops target lesions like these. Erythema multiforme, B like in ‘boy.’ The idea is that erythema multiforme, the target lesions, there are three causes: mycoplasma, herpes, and drugs. So the patient they ask in the exam, the patient has herpes simplex. Herpes simplex is the cause of erythema multiforme in this question. This is what Stevens-Johnson looks like. I just had my own patient. I gave her allopurinol when she got this. Allopurinol can do it, too. Almost died. This is herpes simplex. In a patient who walks in the ICU, if you take a swab out of these sick patients, this will show you multinucleated giant cells. This is also a patient with AIDS who has HSV and did not respond to acyclovir. This is the
Day One

multinuclear giant cells, and then they gave intravenous foscarinet and it cleared up. **So herpes simplex**, the Board loves it. In a different form they'll ask you.
This is No. 2, erythema infectiosum from B-19 parvovirus or Fifth disease, slapped cheek appearance. They ask this complication. It's transient aplastic crisis. If adults get exposed to this virus, they get rheumatoid arthritis-like picture. If I get exposed to this virus, I will not get this erythema infectiosum. I will get arthralgia, which looks typically like rheumatoid arthritis. Adults will get rheumatoid arthritis picture and only transiently, a few days, then it goes away because it's a virus.

This is erythema nodosum, No. 3. The causes of erythema nodosum are in your notes. You notice HIV is not mentioned, so HIV is not a cause of erythema nodosum. What is the most common cause of erythema nodosum in the United States? Sarcoidosis. One of the causes of erythema nodosum is Behçet’s syndrome, and Behçet’s syndrome is painful ulcers in the mouth and genitalia.

So there are three Board type questions. One is erythema nodosum plus diarrhea plus pain in the right lower quadrant. That will be Crohn’s disease. The other is pyoderma gangrenosum plus diarrhea plus pain in the right lower quadrant is also Crohn’s disease, and then erythema nodosum plus non-caseating granuloma means sarcoidosis.

Question No. 4.

An African-American lady has erythema nodosum. What is the next test for diagnosis?

Chest X-ray for sarcoidosis. This is pyoderma gangrenosum. Pyoderma gangrenosum occurs in ulcerative colitis or Crohn’s disease. This is also pyoderma gangrenosum.

Behçet’s syndrome we passed by. We did that. I think my little slides got off. That’s why the problem. In Behçet’s syndrome you get deep and painful ulcers and two of the following: genital ulcers, ocular lesions, skin lesions, or pathergy test. What is pathergy test? Pathergy test is skin hyper-reactivity, any scratches or intradermal saline injection, so skin hyper-reactivity is pathergy test, skin hyper-reactivity. So Question 5 is the one we were talking about diarrhea and pain in the right lower quadrant and the skin lesion of pyoderma gangrenosum.

This goes with No. 7, Question No. 7, common wart. The human papillomavirus is fleshy colored, hyperkeratotic, not umbilicated, and it is painful. This is the one which causes cancer of the cervix, this virus. It can look like this. In the genitalia it could look like condylomata acuminata, same virus.

This is No. 8, umbilicated, painless lesion on the face of a gay man. HIV status was negative three weeks ago. What is the cause of the skin lesion?

[Inaudible].

Poxvirus? It is poxvirus, which is painless, pearly, umbilicated. This is a yearly question, every year the same question, umbilicated mulluscum contagiosum. It is a self-limited disease. Some patients will require treatment; and in the Board exam the answer was cryotherapy, cryotherapy. Cryo therapy. Not crying therapy, cryotherapy. [Laughter] Or you could have crying therapy, too. This is cryotherapy.

No. 9, chicken pox. This is still more or less common. This is chicken pox. Adults get pneumonia and a very high mortality rate. Chicken pox, and in adults you give acyclovir, especially if you’re thinking of pneumonia. There’s very high mortality. Positive Tzanck smear and IV acyclovir or vadarabine.
Question 10, also called Beau’s lines or Mees lines. They are transverse white ridges, transverse white lines. This is seen in arsenic poisoning, arsenic poisoning.

[Inaudible].

Chemotherapy? Chemotherapy is here. Chemotherapy can do it, too.

No. 11, cryoglobulinemia. Hepatitis C, high rheumatoid factor, low complement.

No. 12, hidradenitis suppurativa. This patient uses deodorant. They don’t like to have showers. They just put on deodorant. [Laughter] Staphylococcal infection. The treatment is topical clindamycin, topical clindamycin. In the exam, it is the left axilla. [Laughter] There is no other question of left axilla, only one.

And there is erysipelas. These are very painful cellulitis with raised border due to Streptococcal (sic) pyogenes or Staph aureus. Raised, painful lesions, so this is treated with IV penicillin or cephalaxin. For MRSA you could give clindamycin plus Bactrim. In other words, this is usually treated with cephalaxin or IV penicillin for Streptococcal (sic) pyogenes or Staphylococcal (sic) aureus. Clindamycin plus Bactrim could also be given.

This is hot tub folliculitis due to Pseudomonas, the follicles occur following a bath in the contaminated water, such as hot tubs or spas or water pool, swimming pools. They are typically on the swimsuit area and lesions occur 48 hours after exposure, so you go to a spa and you come home with this. [Laughter]

No. 15.

A 17-year-old female presents with acne on the face. She is concerned that her mother has marks on her face because of acne during her youth. She is very worried about her face getting similar marks. On exam there are comedones on the face that are closed with very little inflammation. What is the best treatment?

This is acne vulgaris. There is very little inflammation, therefore you could just treat with Tretinoin cream. C is the answer. If there was inflammation, you would also use benzoyl peroxide gel. There are comedones with keratin plugs.

Question 16, a 16-year-old patient is brought by her mom to assess need for Accutane. On exam there are no cysts or nodules, only a few open and closed comedones with significant inflammation. What is the treatment?

So in this case a significant inflammation, therefore the answer is topical benzoyl peroxide gel. You shall see the next page. It describes that, the treatment. If there is inflammation, you give benzoyl peroxide. If there is no inflammation, Tretinoin, or you could give both. If there is inflammation also, give Tretinoin at nighttime, benzoyl peroxide in the morning.

The most important question is on tinea versicolor. Tinea versicolor is a fungus which gives lesions that are scaly, circular, light brown or hypopigmented macules, and they are always on the exam. Treatment is with antifungal medication. They usually occur at the time of high humidity. KOH preparation will show short hyphae and round spores. They call it spaghetti and meatballs. When you read the sentence ‘spaghetti and meatballs’ in the exam, that means tinea versicolor. The treatment is solutions containing sulfur, salicylic acid, selenium sulfide, or antifungal medication. This is a very common disorder. This is what is called spaghetti and meatballs.
No. 18 is tinea corporis. Tinea corporis again. Again a fungal infection, superficial. Tinea corporis on the body causing erythematous, annular, scaly plaque with central clearing, just like in Lyme disease. This is tinea cruris, ringworm. This is ringworm, tinea cruris. The margins are scraped to check for fungal hyphae.

No. 19.

A woman notices a 45-year-old coworker scratching her head repeatedly while at work. She gives you a history of taking hormone therapy for menopause. On exam there is an area of distinct patch of hair loss on her with erythema, scaly skin, and hair loss near the right temporal area. When tested with a Wood's lamp, it is positive. You see bright green fluorescence of the infected hair. What is the diagnosis?

Tinea capitis. You have to treat anybody who is associated with her, black dots are there, broken hair, some plaques with pustules, they’re called karyon, this is a ringworm of the scalp.

The next one is actually alopecia areata, Question No. 20.

Sudden loss of hair occurs from the scalp of a 12-year-old patient during a period of two weeks. Areas of alopecia are smooth, circular, and discrete with complete hair loss. What is the cause?

A patient with alopecia areata typically has smooth, circular, discrete area of complete hair loss that develops in a period of a few weeks with the regrowth over several months. There’s always regrowth. Whenever the tension goes away, it comes back. In other words, the disease goes away when the tension goes away. This is not nervous hair pulling, which can also occur, or traction alopecia. That is different. That's this one. The answer is B like in 'boy' for Question 20.

Question 21, a 40-year-old male presents with scaly, itchy rash. Itching is more prominent at night. On exam there are pustules and vesicles on the scrotum, penile shaft, hands, feet, including the interphalangeal clefts. Treatment?

This question is always on the exam. The answer is 5% permethrin, 5% permethrin. It is scabies. It is distributed on these areas on the body, specifically in the interphalangeal area, very itchy lesions, and 5% permethrin is the treatment. It is an infestation of the skin by the mite of scabies that results in intense pruritic eruption with a characteristic distribution pattern as shown here.

Question 22.

A 31-year-old man presents with nonspecific complaints of arthralgia and fatigue. He thinks he is working very hard because of a tough work schedule. He gives a past history of non-gonococcal urethritis and Hepatitis B. A picture of his face is shown with scaly skin on the chin and around the moustache. Which of the following is associated with this lesion?

This question was asked last year. The answer is A, positive HIV. I don't know why they put patient has tough work schedule. [Laughter] The patient has tough work schedule? It’s due to HIV. [Laughter] The treatment of this condition is in your notes. Low potency corticosteroids are used daily until improvement is seen. That is, you have to treat HIV also. That's beside that. A lot of people have this without HIV. This is also seborrheic dermatitis. That is seborrheic dermatitis. This is seborrheic dermatitis.
This is *Candida*, 23. *Candida* occurs in intertriginous areas, tongue, esophagus, vagina, diaper’s rash in babies, like this one. Treatment is antifungal medications. This is *Candida* or geographical tongue?

**Candida. [in unison]**

This is geographical tongue, okay, so this is all *Candida*, this from here on, *Candida*, and treatment of *Candida* is what? Fluconazole.

No. 24.

A construction worker with alcohol on his breath presents with a vesicular, itchy rash on the palms and hypertrichosis. He is not on any medications. What is the diagnosis?

This is bullous lesion, itchy, hypertrichosis. The urine is red in the morning.

**Porphyria.**

Porphyria. Hepatitis C. Porphyria in one picture, hyperpigmentation, hypertrichosis, bullous lesion. Treatment is in your notes.

No. 25. Before you go to that, porphyria cutanea tarda treatment also in the notes, also on the slide. Dermatitis herpetiformis, celiac disease, very itchy, associated with celiac disease. Treatment is gluten-free diet plus or minus Dapsone. D-a-p-s-o-n.

Next, No. 26, keratoderma blennorrhagicum of Reiter’s syndrome. Reiter’s. These patients are young men complaining of pain in the heel. They have circinate balanitis like this one, painless erosions, circinate balanitis, painless erosions of Reiter’s.

No. 27 is always on the exam.

A 16-year-old female who’s sexually active goes to Connecticut for camping. There she happens to have sex with the counselor. She now comes to you because of an itchy erythematous patch on her trunk and spreading to proximal extremities. She denies a tick bite. What is the diagnosis?

She has a herald patch here. They’re showing the herald patch which always occurs on the chest, even in children. It occurs on the chest, herald patch. I show you here. This is a round patch of pityriasis rosea, and in the exam question she got that after sex with the counselor. It is a virus due to picornavirus, and the patch will come first on the chest area, usually along the rib lines, followed by a widespread, bilaterally symmetrical—this is the patch—followed by widespread, symmetrical itchy skin eruptions on the trunk, on the upper extremities. And the center of the lesion is crinkled or like cigarette paper, cigarette paper in the center, and you notice one thing. The distal part of the extremities are not involved. Distal parts are free of the lesions, and that is very characteristic of pityriasis rosea, the patch on the chest followed by lesions all over, except the distal part of the extremities. It is a picornavirus, pityriasis rosea, always on the exam, so the moral of the story is?

*[Inaudible]*.

Sometimes they ask the treatment. The treatment is topical steroid cream, which is true for every skin condition, as far as I’m concerned. [Laughter] Actually, I carry steroids in my pocket all the time. Any skin condition, I just give steroids. [Laughter] No. 28, the treatment of the above condition, we just did. That is the one, topical steroid cream.
No. 29.

A lady is presented with facial rash that gets worse with sun exposure and alcohol. There are no comedones. Treatment of choice is?

[Inaudible].

This is rosacea getting worse with the sun and alcohol, rosacea. Rosacea can look like this or this or this, and the treatment is local metronidazole or [inaudible] cream. They're showing you before and after topical metronidazole. This was also on the exam.

This is No. 30.

A 64-year-old man presents with velvety hyperpigmented plaques in intertriginous areas, dark patches in the axillae, and in the left groin. It is associated with?

[inaudible].

Board question. This is associated with cancer of the stomach. This is called acanthosis nigricans. Acanthosis nigricans occurs in insulin resistance, in diabetes mellitus. Again, causes of acanthosis are insulin resistance, diabetes mellitus, malignancy of the stomach, and there's one more. There's no mention here, but I know it is polycystic ovarian disease. We'll do that on endocrinology again and also acromegaly.

No. 31.

In the development of skin cancer, which of the following is most important?

How many people have A? One, two, three, four, five, six. B? And the rest will be C, I guess. I'm glad you came here, all of you, [Laughter] because the answer is A. Very few said it was A. Very few were A. The answer is A, and you should be happy that you came because this is a board question. If you were exposed in childhood, severe sun exposure, you will get, you will have very high chance of cancer of the skin later on in life. That is written in the literature everyplace.

Next is melanoma, which looks like this, vicious-looking, nodular lesion, and the precancerous lesion is dysplastic nevus. This is a precursor lesion for melanoma, dysplastic nevus. In the middle of it you can see the melanoma coming on. Fifty percent of people having dysplastic nevus, 50% of dysplastic nevi will develop melanoma like this one, and the Board question is on prognosis. Prognosis in Stage 1 is the best where the depth of the lesion is less than .06mm. The survival is more than 95%, ten years. So there are two questions on melanoma you should know. One is what is the best prognosis? That means the depth of the lesion is less than 0.76mm, and the second Board type question is not here, but it will come again in oncology, but I can tell you right now that it is a melanoma was removed in one patient and there were lymph nodes present, and the answer was after you remove the lymph nodes, the treatment is radiation or chemo, they asked. The answer was chemotherapy with high-dose interferon, so, in other words, once you remove melanoma, which is a nodal disease which is Stage 3 in this case, you will then give chemotherapy with high-dose interferon. That was the second question, so there are two questions. We'll do them again in oncology just to be sure that you know about it.

This is squamous cell carcinoma of the skin, which is also related to sunlight and cigarettes. Sunlight and cigarettes are the risk factors, and the precursor lesion of squamous cell carcinoma of the skin is actinic keratosis. This is actinic keratosis which people get with sun
exposure. It leads to this or this. These are hyperkeratotic papules on the sun exposed areas, and they have a high metastatic potential, high metastatic potential in squamous cell carcinoma.

Compare that with the next one, which is—this is [inaudible] keratosis, causes squamous cell cancer. Compare that with this one, which is basal cell carcinoma, the next one. Basal cell carcinoma is much less malignant. It has clearly defined margins. It is shiny, pearly, translucent quality with telangiectasias on it. Sun exposure is also a risk factor, but its metastatic potential is lower than that of squamous cell cancer, so it is not as bad as this one is. This one is bad. This is bad. But, this is not that bad, but it is also cancer of the skin.

The one which you should know for the exam purpose is No. 35, an elderly man with several months of skin eruptions, usually on the back in the examination, mycosis fungoides. It is the most common cutaneous T-cell lymphoma, always in the exam. An older person who has this lesion for many, many months, sometimes visceral involvement will occur with a five-year survival of 60%. Mycosis fungoides is a favorite of the Board.

Last, this one, No. 36. All of the following are associated with vitiligo except? This is vitiligo. Which one of these is not associated with vitiligo? Because vitiligo is associated with many autoimmune disorders. The answer is H. H is not associated with it. Everything else is. It is a part of polyglandular deficiency syndrome, which we'll do again in endocrinology. There are new questions. The answer is H, except H. Everything else is associated with vitiligo. H is Cushing's disease. It is not associated.

Okay, Board questions.

What is the premalignant precursor factor for squamous cell carcinoma of the skin?

You just did it. Actinic keratosis. Next?

A 41-year-old healthy lady gardener has a cat at home. She has had three recurrent episodes of armpit lumps that drain serous fluid. There is no fever. She has been using a new deodorant under her armpits. The rest of the exam is normal. What is the diagnosis?

New deodorant?

Hidradenitis.

Next.

A 51-year-old female presents because of many warty, well-circumscribed, scaly and hyperpigmented lesions on her trunk. On exam the lesions have a 'stuck-on' appearance. A biopsy will reveal which of the following?

[Inaudible].

This is called—the answer is A. It is seborrheic keratosis, and this is what it looks like, a 'stuck-on' appearance, that something is stuck on the skin, you want to take it off. Okay? 'Stuck-on' appearance. So I have mentioned about it under the question. You read about it because it has some associations with a variety of malignancies, including GI and lung cancer.

No. 4.

A 62-year-old patient with weight loss and abdominal fullness develops seborrheic keratosis. Which of the following is your next step?
Seborrheic keratosis. That is the one we just did, which is associated with cancers, so therefore you will do CAT scan of the abdomen for GI malignancy. GI malignancy is associated with seborrheic keratosis. The answer is B like in 'boy.'

Ladies and gentlemen, thank you very much. I will see you tomorrow morning at 9:00 exactly. Thank you.

[End Day 1]